

# The epidemiology of sleep apnoea

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## Introductory article

### A community study of snoring and sleep-disordered breathing: prevalence

LG Olson, MT King, MJ Hensley, NA Saunders

*We conducted a study of the prevalence of sleep-disordered breathing in subjects derived from a random sample of the population. A total of 2,202 subjects 35 to 69 yr of age were approached. Four hundred forty-one answered a questionnaire concerning their sleep symptoms, general health, and habits such as alcohol consumption, and they were monitored for sleep-disordered breathing (SDB). The sample was biased in favor of snorers and those with other subjective sleep complaints. Fifty-six percent of the subjects were men. Of the 441 subjects 79 (17.9%) had SDB (more than 15 episodes of apnea or hypopnea per hour: respiratory distress index [RDI]  $\geq 15$ ), 289 were snorers but had RDI  $< 15$ , and 73 were nonsnorers. The prevalence of SDB in this sample was therefore at least 3.6% (79 of 2,204). The minimum prevalence in men was 5.7%, and in women it was 1.2%. Logistic regression identified only male sex as an independent predictor of snoring without SDB (adjusted odds ratio [OR], 3.24; 95% CI, 1.33 to 7.82), body mass index (adjusted OR for an increase of 5 kg/m<sup>2</sup>, 0.95; 95% CI, 0.85 to 1.05), and alcohol consumption (adjusted OR for an increase of 10 g/day, 1.05; 95% CI, 0.84 to 1.37) were not significant predictors of snoring. The independent predictors of SDB among snorers were age (adjusted OR for an increase of 5 yr, 1.26; 95% CI, 1.08 to 1.47) and neck circumference (adjusted OR for an increase of 2 cm, 1.53; 95% CI, 1.16 to 2.00). Alcohol consumption was not a factor (adjusted OR for an increase of 10 g/day, 0.98; 95% CI, 0.86 to 1.12). (Am J Respir Crit Care Med 1995;152: 711-6)*

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The study by Olson *et al* is the latest in a series of large studies which describe the community prevalence of sleep disordered breathing. The results of this study are presented in three papers,<sup>1-3</sup> the introductory article being the one which addresses prevalence. This study, together with several other similar studies, attempts to define the community frequency of abnormal breathing during sleep. However, there are still important questions about the apparently widely differing prevalences in these studies and the overall significance of these figures for the understanding of obstructive sleep apnoea and its health care ramifications. This commentary deals with these issues.

#### How common is disturbed breathing during sleep?

The first and superficially simplest question concerns the prevalence of disturbed breathing during sleep in the normal population. There are now at least 12 large studies of adults from seven countries and four continents addressing this question (table).<sup>2-13</sup> That of Olson *et al*<sup>2</sup> from Newcastle, Australia is the most recent. The apparent disease prevalence reported by these studies varies from as little as 0.3% to as much as 15% (table). The Oxford prevalence study<sup>10</sup> and the study by Olson *et al*<sup>2</sup> lie towards opposite ends of this spectrum. In

practice, the results of these studies are more in agreement than it first appears – it is not true that sleep apnoea is about 50 times more common in Australia than it is in Oxford! To illustrate this point, the Oxford results will be reanalysed and compared with those reported by Olson *et al* in the introductory article. Some of the data corrections presented during this re-analysis are necessarily approximate and the calculated prevalences are therefore also probably inexact and should be interpreted in this light.

The definition of what constitutes significant sleep apnoea is critical in these studies. In any large population sample the severity of sleeping respiratory disturbance is unimodally distributed, there being no discrete subgroup who “have the disease”. Hence, the selection of a single diagnostic cut-off point above which breathing is “abnormal” is misleading. Figure 1A shows the population distribution of nocturnal hypoxaemic dipping (which indicates transient hypoventilation or apnoea) in the 893 randomly selected normal men from the community studied in the Oxford prevalence study.<sup>10</sup> Clearly it makes a large difference to the apparent disease prevalence whether a threshold of  $>5$  or  $>10$  events per hour is used to indicate abnormality. Quite correctly, therefore, Olson *et al*<sup>2</sup> report their prevalence results as frequencies at a number of different severity

Details of the 12 major prevalence studies of obstructive sleep apnoea							
Author	Reference	Country	Population	Age range	Method	Criteria	Estimated whole population prevalence
Franceschi <i>et al</i> (1982)	4	Italy	2518 consecutive hospital admissions (M and F)	6–92	1) Questionnaire 2) Polysomnography on subgroup of 87 (no oximetry)	AHI $\geq$ 10/hour	1%
Lavie (1983)	5	Israel	1262 M	Working age	1) Questionnaire 2) Polysomnography on 78 (no oximetry)	AHI $\geq$ 10/hour AHI $\geq$ 20/hour	2.7% 0.7%
Berry <i>et al</i> (1986)	6	USA	60 heavy snorers	30–?	1) Newspaper advert recruitment 2) Polysomnography on 46	AHI $\geq$ 5/hour	?1.3%
Telakivi <i>et al</i> (1987)	7	Finland	Approximately 278 M	41–50	1) Part of a twin study 2) 25 snorers and 27 non-snorers had polysomnography	AHI $\geq$ 20/hour >10/hour $\geq$ 4% Sao <sub>2</sub> dips	0.4% 1.4%
Gislason <i>et al</i> (1988)	8	Sweden	3100 M	30–69	1) Questionnaire 2) 61 sleepy snorers had polysomnography	AHI $\geq$ 10/hour AHI $\geq$ 5/hour	0.9% 1.4%
Cirignotta <i>et al</i> and Lugaresi <i>et al</i> (1989)	9 and 38	Italy	1170 M	30–69	1) Questionnaire 2) 40 heavy snorers had polysomnography	Severe and symptomatic AHI $\geq$ 10/hour AHI $\geq$ 5/hour	0.5% 3.3% 5.1%
Stradling and Crosby (1991)	10	Oxford, UK	1001 M	35–65	Oximetry on 893	Severe, symptomatic and >20/hour $\geq$ 4% Sao <sub>2</sub> dips (>3%) >10/hour $\geq$ 4% Sao <sub>2</sub> dips (>3%) >5/hour $\geq$ 4% Sao <sub>2</sub> dips (>3%)	0.3(1)% 1(3)% 5(14)%
Gleadhill <i>et al</i> (1991)	11	Northern Ireland	920 M	40–64	1) Questionnaire 2) Oximetry on 138 "symptomatic" and 164 "controls"	>10/hour $\geq$ 3% Sao <sub>2</sub> dips + symptoms	0.8%
Young <i>et al</i> (1993)	12	Wisconsin, USA	1670 M 1843 F	30–60	1) Questionnaire 2) 602 had polysomnography	AHI $\geq$ 15/hour AHI $\geq$ 10/hour AHI $\geq$ 5/hour AHI $\geq$ 15/hour AHI $\geq$ 10/hour AHI $\geq$ 5/hour	9% 15% 24% 4% 5% 9%
Olson <i>et al</i> (1995)	2	Australia	1233 M 969 F	35–69	1) Questionnaire 2) Biased subset of 441 had respiratory measurements	AHI $\geq$ 15/hour AHI $\geq$ 10/hour AHI $\geq$ 5/hour	4–18% 7–35% 14–69%
Bearpark <i>et al</i> (1995)	13	Australia	486 M	40–65	1) Questionnaire 2) 311 had oximetry, snoring and heart rate	>10/hour $\geq$ 3% Sao <sub>2</sub> dips + snoring/pulse rise >5/hour $\geq$ 3% Sao <sub>2</sub> dips + snoring/pulse	10% 26% with symptoms
Kripke <i>et al</i> (1995)	Personal communication	San Diego, USA	165 M 190 F	40–64	Telephone recruitment. 1467 reached, 1085 interviewed. 417 visited at home, 355 studied (oximetry and actigraphy – 3 nights)	>20/hour $\geq$ 4% Sao <sub>2</sub> dips (men) >20/hour $\geq$ 4% Sao <sub>2</sub> dips (women) >20/hour $\geq$ 4% Sao <sub>2</sub> dips (combined)	9% 5% Whites 5%, non-whites 16%

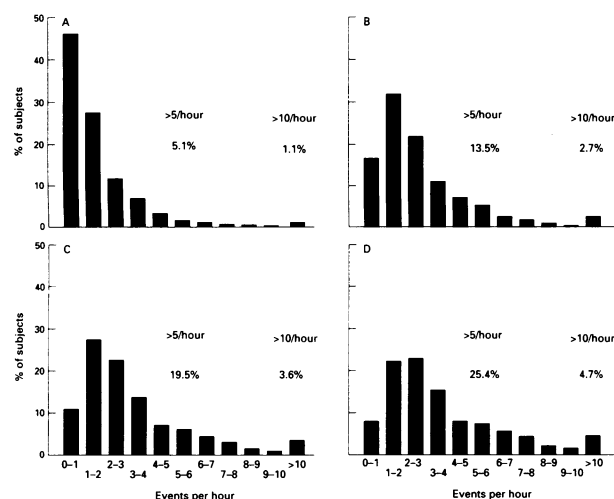
AHI = apnoea/hypopnoea index; Sao<sub>2</sub> = arterial oxygen saturation

thresholds. From these results the reported population prevalence of "sleep apnoea" varies between 13.8% and 7% in Australia (men and women) and 5.1% and 1.1% in Oxford (men only), depending on whether >5 or >10 events per hour is used as the cut-off point.

The second complexity in comparing these studies relates to the methods used to quantify sleep apnoea. Differences in equipment and the processing of signals can substantially change the apparent prevalences. In the Oxford study the stored data from the Biox 3700 pulse oximeter were used and an abnormal event was defined as a saturation fall of  $\geq$  4% from the previous high. Since this oximeter does not resolve the oxygen saturation to less than 1% in this mode, this equates to a minimum fall of 4–5%. Reducing this threshold by just 1% substantially increases the apparent prevalence

of the breathing disturbance. The storage algorithm of this oximeter also reduces the number of documented events by a small percentage.<sup>14</sup> The effects of changing the event threshold to  $\geq$  3% and correcting for the storage algorithm are shown in fig 1B. This results in a rise in the apparent prevalence of "sleep apnoea" in the Oxford study to 13.5% at >5 events per hour and 2.7% at >10 events per hour.

Studies based on arterial oximetry<sup>10 11</sup> (and Kripke, personal communication) generally produce lower prevalence figures than those based on detailed recordings of breath cessation and respiratory movement (including the study from Olson *et al*<sup>2</sup>). This is because some events fulfil the standard criteria for abnormality in terms of respiratory movement and airflow but do not cause sufficient arterial desaturation to fulfil the usual



**Figure 1** Calculated effects of changes in methodology and population characteristics on the prevalence of nocturnal hypoxaemic dipping in normal men drawn from an Oxfordshire village. The percentage of subjects above two arbitrary thresholds for abnormality (>5 and >10 events per hour) are shown for each analysis. (A) Prevalence of  $\geq 4\%$  oxygen saturation dips in 893 normal men.<sup>10</sup> (B) Effect of changing the definition of an oxygen saturation dip to  $\geq 3\%$ . (C) Correction of the oximetry results for the estimated "under reporting" of abnormalities by oximetry compared with the apnoea/hypopnoea index (AHI), see text. (D) Estimated effect of correcting the Oxford results for the level of obesity in the USA.

desaturation criteria.<sup>15</sup> This is, of course, entirely a problem of definition. It means that an average subject in the community with 15 events per hour using an oximetry criterion of  $\geq 4\%$  saturation dips will be "worse" than an average subject with 15 events per hour using standard breathing criteria, and that sleep apnoea is apparently more common when studied using breathing criteria. Douglas *et al*<sup>15</sup> from Edinburgh have shown that, on average, using  $\geq 4\%$  oximetry dips detects about 20% fewer events than conventional breathing criteria. Figure 1C presents the Oxford community survey data corrected for this 20% "under reporting" – a further apparent increase in prevalence is seen.

The next issue that must be considered when comparing studies is the genuine differences in disease prevalence between different populations due to differing degrees of obesity. Obesity, particularly neck obesity,<sup>16</sup> is the most important adult risk factor for sleep apnoea, and populations where average body weight is higher (or where fat deposition tends to favour the upper body such as Asian groups<sup>17</sup>) will therefore have higher disease prevalences. In the USA and Australia average body mass indices exceed those in the UK and, not surprisingly, there are higher disease prevalences in these populations.<sup>2,10-12</sup> Figure 1D shows the increase in prevalence which might have been expected in the Oxford survey if the studied population had shown the same obesity profile as that seen in Wisconsin, USA where the largest respiratory signal based survey was performed.<sup>12</sup> The Oxford prevalence now apparently exceeds the minimum prevalence reported by Olson *et al*<sup>2</sup> despite the apparently substantially lower figure in the original paper (table).

Methodological differences in the selection of subjects also make comparison of different studies difficult. Reports which aim to study the whole population tend to use simple assessment methods because of the considerable number of subjects involved. Those which have used traditional complex sleep laboratory methods

could not study the whole population and so selected only a small subset and referred back to the original population to estimate a minimum prevalence (table). Selection of the subset for sleep study has usually been by intentional identification of a sample at high risk (often by questionnaire) or by random selection. Olson *et al*<sup>2</sup> adopted the latter approach and eventually performed technically adequate sleep studies on 441 of 2202 (20%) of their originally contacted subjects. This is comparable with the study of Young *et al*<sup>12</sup> who performed technically adequate detailed laboratory sleep studies on 605 of 4284 (14%) of an initially contacted group, and substantially less than 893 of 1001 (89%) studied with oximetry alone in the Oxford study.<sup>10</sup> The selection of a small subset for sleep study inevitably introduces the risk of bias, and the smaller the subset, the greater the risk is likely to be. Problems of this nature afflict the Olson study and are recognised by the authors.<sup>2</sup> Their original study population from which they drew their subset for sleep studies was not truly random (half were volunteers for a coronary risk study and these included a considerable excess of snorers), and there were considerable response biases among those who volunteered to participate. These biases combined to produce too many men (1.54 times the number expected), snorers (1.24 times expected), and subjects who considered themselves to have a sleep problem (2.4 times expected) among those who underwent a sleep study. Olson *et al* are unusual in having identified possible sources of bias and this laudable strength in their study much improves its interpretation. Bias is probably the explanation for the extremely high prevalence of sleep disordered breathing in the subset who had sleep studies. Over 35% of those studied showed more than 10 respiratory events per hour and >17% had 15 per hour (commonly accepted thresholds for significant abnormality). Over 8.5% showed >25 per hour – substantial abnormality by any criterion. Given these biases, it is sensible to interpret these findings conservatively. The most cautious approach is to assume that all the subjects in the original population with sleeping respiratory abnormalities were included in the subset who underwent sleep studies. Using this highly cautious approach, minimum population prevalences of 1.72% with >25 events per hour and 3.58% with >15 per hour are identified.

Some recent data on the milder variants of sleep apnoea suggest that even these high figures may *understate* the genuine prevalence of respiratory disturbances to sleep. Heavy snoring alone can fragment sleep without requiring frank apnoea, hypopnoea, or arterial oxygen desaturation.<sup>19</sup> This phenomenon would not have been detected by any of the studies to date. Sleep fragmentation is the most important feature of obstructive sleep apnoea in causing symptoms<sup>20</sup> and so there may exist some further treatable respiratory sleep disturbance which is due to heavy snoring alone.<sup>19</sup>

#### Is all community sleep apnoea obstructive?

The startlingly high prevalence of occult obstructive sleep apnoea in the community has led some observers to question whether all or most of this abnormality is truly obstructive in aetiology. If not, then its pathological significance would be in doubt and its treatment responsiveness uncertain.

Olson *et al*<sup>2</sup> intentionally selected their subset for sleep study to include a relative excess of older subjects in order to assess the prevalence of abnormality over a large age range. In the elderly central sleep apnoea

becomes very common,<sup>21,22</sup> probably due mainly to sleeping periodic breathing associated with cerebrovascular disease, heart failure, and a reduced sleep depth. The high prevalence of sleep disordered breathing reported by Olson *et al* in subjects aged 65–69, of whom 40% showed >15 respiratory events per hour, may partly be explained by this. However, most of the excess of periodic breathing in the elderly is seen in people aged over 70 and Olson *et al*<sup>2</sup> did not study patients as old as this. In younger patients non-obstructive causes of sleep apnoea represent <1% of the abnormal cases seen in a sleep laboratory,<sup>15</sup> and clinically well subjects with significant central sleep apnoea are virtually unknown. Indeed, so uncommon is truly central sleep apnoea in well subjects from younger age groups that the majority of subjects with “central sleep apnoea” by polysomnographic criteria actually have primarily obstructive apnoea with reflex suppression of respiratory effort secondary to their pharyngeal collapse.<sup>23</sup> It is therefore most unlikely that many cases of undiagnosed truly central sleep apnoea exist in the community to distort the results of prevalence studies, but which escape detection in sleep laboratories.

Another approach to this question is to assess whether the numerous subjects with disordered breathing during sleep also have the other symptoms which would be expected if their breathing abnormality is primarily obstructive. Olson *et al*<sup>1</sup> have specifically addressed this question. They found that the usual clinical features of pharyngeal airway obstruction (snoring, partner witnessed apnoeas, gasping or choking sounds during sleep, and restless sleep) were substantially more common in their subjects with proven sleep apnoea than in those without. Of the subjects shown to have sleep disordered breathing, 71% reported disruptive snoring while only 19% of those found to be normal were reported to snore at home.

It therefore seems clear from what is known of the pattern of sleep disordered breathing seen in sleep laboratories and from the symptom characteristics of the people in the community prevalence studies, that the overwhelming majority of the sleep disordered breathing identified in young people from the community is due to obstructive sleep apnoea and its variants.

### Does all obstructive sleep apnoea need nCPAP treatment?

The finding that 1–5% of the adult male population has the obstructive sleep apnoea syndrome (appreciable sleeping respiratory disturbance and some daytime sleepiness) is striking. Do one in 20 men need life long nasal continuous positive airway pressure (nCPAP)? The awareness, but poor understanding, of this question is currently tending to provoke an inappropriate “denial” response from those responsible for funding health care. This response risks the refusal of highly effective treatment<sup>24</sup> to patients with symptomatically unpleasant disease which poses a mortality risk to themselves (through respiratory failure, etc<sup>25</sup>) and others (through sleepiness related road accidents, etc<sup>25,26</sup>). A more rational perception of the problem is needed.

The treatment of obstructive sleep apnoea is aimed primarily at controlling the complications of the disease (excessive daytime somnolence, respiratory failure,<sup>25,26</sup> etc) rather than the respiratory disturbance itself. It remains unclear to what extent the many subjects in the community with objective respiratory disturbance during sleep suffer these complications. For all diseases, patients attending a hospital clinic will represent those

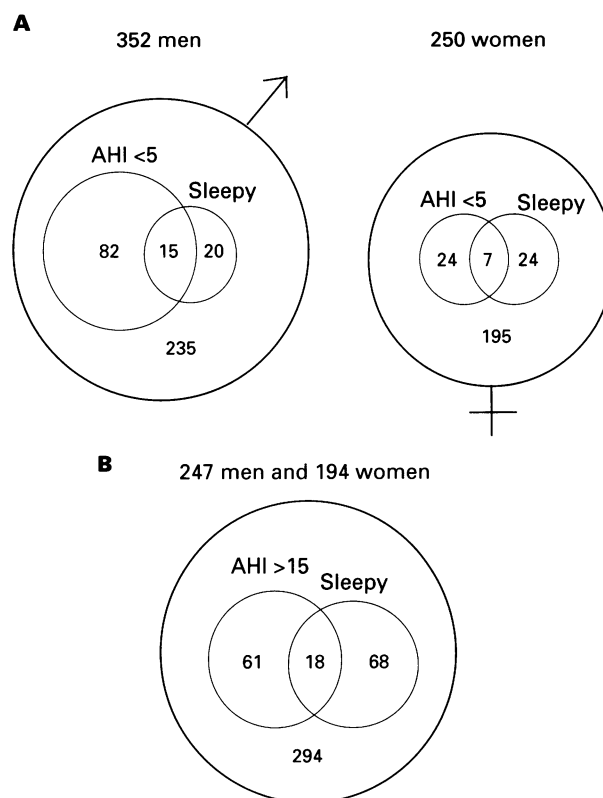


Figure 2 Interrelationships between objective nocturnal respiratory disturbance and self reported daytime excessive sleepiness in subjects who had sleep studies in the two largest breathing signal based prevalence studies: (A) Young *et al*;<sup>12</sup> (B) Olson *et al*.<sup>2</sup> In neither of these analyses is the excess of hypersomnolent subjects in the group with objective breathing abnormality statistically significant ( $\chi^2$  test).

particularly disabled by their disease. Hospital asthma and diabetic clinics convey the impression that these common diseases are usually a major burden for their sufferers. In reality, in the community this is far from the truth. More than 10% of the population have some degree of asthma<sup>27</sup> and >1% have diabetes,<sup>28</sup> but most of these people pursue active lives and require only simple treatment.

The medical management of other common diseases with a wide spectrum of severity (asthma, diabetes, hypertension, etc) is based on a flexible and staged therapeutic response. In asthma such an approach is considered so appropriate that it has been established through guidelines.<sup>18</sup> Unfortunately, until recently there have only been two effective but arduous treatments for adult obstructive sleep apnoea – nCPAP and tracheostomy (with a minor role for tonsillectomy). Treatment for this disease has not been sufficiently sophisticated to allow a “flexible response” strategy. A population prevalence of 5% for the sleep apnoea syndrome is therefore inappropriately equated with one in 20 men requiring nCPAP. In reality this is no more likely than a >10% prevalence of asthma in the community requiring that one in 10 adults has oral steroids and a nebuliser. New simpler treatments for mild to moderate obstructive sleep apnoea, such as removable mandibular advancement splints,<sup>29</sup> are now being developed and these should lead to a more flexible treatment approach which is tailored to the individual needs of patients.

The main indication for the treatment of sleep apnoea is excessive daytime sleepiness since it is disabling and responds well to appropriate treatment.<sup>24,25</sup> The relationship between daytime sleepiness and the objective

## LEARNING POINTS

- \* 1–5% of adult men from normal communities have the “sleep apnoea syndrome” (objective sleeping respiratory disturbance associated with daytime sleepiness).
- \* The prevalence in normal communities increases with the level of obesity.
- \* The considerable differences in reported prevalence between different studies are probably largely attributable to differences in disease definition and measurement.
- \* Obstructive sleep apnoea is a common disease with a wide spectrum of severity (much like asthma and diabetes) and so its management should be flexible and staged, with different treatments being used depending on symptom severity, complications, and objective respiratory abnormalities.

severity of obstructive sleep apnoea is complex. Even within a sleep clinic population, where particularly sleepy patients tend to collect, the relationship between these variables is loose.<sup>30,31</sup> This is likely to be because the traditional indices of sleep apnoea severity do not assess the associated sleep fragmentation very well, or because sleepiness occurs when sleep apnoea is associated with one or more co-factors – much like ventilatory failure in obstructive sleep apnoea which selectively occurs when there is coincident severe obesity, mild airflow obstruction, or muscle weakness.<sup>32</sup> The problems with the current assessment of sleep fragmentation are illustrated by work on the evolution of arousal which shows that the patterns of the cortical electroencephalogram during obstructive sleep apnoea are complex<sup>33</sup> and some “arousal” events, detectable by induced autonomic changes, can occur without any EEG changes at all.<sup>34</sup>

The estimate that 1–5% of the male population has the “sleep apnoea syndrome” may be an overestimate of true community prevalence since some of these hypersomnolent subjects in the community with sleep apnoea will have a proportion of their sleepiness explained by non-respiratory factors. Figure 2 shows the inter-relationships between objective breathing abnormalities during sleep and subjective daytime sleepiness calculated from the two largest sleep apnoea prevalence studies based on breathing criteria assessments.<sup>2,12</sup> From these calculations the frequency of subjects reporting themselves to be sleepy in the subgroup with an objective nocturnal breathing abnormality is not statistically significantly increased compared with the parent population. This emphasises that, despite the obvious severe and treatment responsive sleepiness frequently seen in patients attending a hospital clinic,<sup>24,25</sup> in the general population much of the community burden of hypersomnolence is attributable to non-respiratory factors.

The other main reason advanced for the treatment of obstructive sleep apnoea is the reduction of an associated cardiovascular disease risk. There is no doubt that patients with obstructive sleep apnoea show an excess of hypertension, coronary heart disease, stroke, and sudden death.<sup>25</sup> It remains unclear, however, whether this is directly due to their sleep apnoea or the confounding cardiovascular risk factors which co-correlate with sleep apnoea, particularly obesity.<sup>25</sup> This problem – and many of the methodological difficulties which make its resolution difficult – are well illustrated in the study by Olson *et al.*<sup>3</sup> They found clear associations between sleep apnoea and hypertension, coronary heart disease, and occlusive vascular disease, but these could be explained by the confounding effects of age, sex, obesity, smoking, and alcohol intake. A full discussion

of the issues relating to the risk of vascular disease is inappropriate here and is fully explored elsewhere.<sup>25,35,36</sup> Although sleep apnoea is associated with dramatic nocturnal haemodynamic instability and an overall nocturnal rise in blood pressure,<sup>37</sup> it remains unclear how these events relate to daytime vascular consequences.

## Conclusions

The literature on the prevalence of obstructive sleep apnoea is superficially complex and conflicting. However, the actual situation is probably clearer than the literature suggests. When methodological and disease definition factors are considered, about 1–5% of adult men, and rather fewer women, have the “sleep apnoea syndrome” (objective breathing abnormalities during sleep associated with daytime sleepiness). Due to population differences in obesity, this syndrome is more common in the USA and Australia than it is in the UK. Like non-insulin dependent diabetes and asthma, obstructive sleep apnoea is an extremely common disease in which stratified therapy is required. This needs to be adjusted to the patient's symptoms and disease complications and not just based on simple study criteria. The evolution of services and treatment techniques to manage this disorder appropriately must continue since the epidemiological studies have now shown that the prevalence of this problem is too great to ignore.

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